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Causes of Stability of Aggression from Early Childhood to Adolescence: A Longitudinal Genetic Analysis in Dutch Twins

C. E. M. van Beijsterveldt,[1,3] M. Bartels,[1] J. J. Hudziak,[2] and D. I. Boomsma[1]

This study investigated the contribution of genetic and environmental influences on the stability of aggressive behavior from early childhood to adolescence. Two developmental models, the simplex model and the common factor model, were tested to study the underlying processes of stability and change. Measures of aggressive behavior (AGG) were obtained from maternal CBCL data as part of a large ongoing longitudinal study of the Netherlands Twin Registers (NTR) and included data from 6488 three-year-old twin pairs, 5475 seven-year-old twin pairs, 2983 ten-year-old twin pairs, and 1509 twelve-year-old twin pairs. AGG showed moderate to high stability during childhood. The stability coefficients ranged from 0.41 to 0.77 across varying intervals. Averaged across boys and girls, genetic factors accounted for approximately 65% of the total stability. Longitudinal genetic analysis indicated a simplex model for genetic effects, which suggests a dynamic development process consisting of transmission of existing genetic effects interacting with new genetic influences. This is especially true at age 7, when the influence of new genetic factors was large. Shared environmental factors accounted for approximately 25% of phenotypic stability, and it seemed that a stable set of the same shared environmental factors underlay the development of AGG. Nonshared environmental factors, when important, are age specific. Sex-specific differences for stability were identified. For boys, genetic influences were greater, whereas for girls shared environmental factors were more important. These data support the idea that both genetic and environmental influences play a role in the stability of AGG from age 3 to 12.

KEY WORDS: Aggressive behavior; longitudinal; heritability; childhood; twins; CBCL.

INTRODUCTION

Aggressive behavior (AGG) in childhood is a stable behavioral trait that persists to a considerable degree into adulthood. For example, Verhulst and van der Ende (1995) investigated the developmental course of problem behaviors across 2- to 8-year time intervals in Dutch children and reported high stability of aggressive behavior, with correlations of 0.65, 0.60, 0.52, and 0.48 for, respectively, 2-, 4-, 6-, and 8-year intervals for boys ages 4–6 years at the first assessment. Over a 14-year period these stability correlations for aggressive behavior were 0.53 for females and 0.33 for boys (Hofstra *et al.,* 2000). Although these studies revealed considerable stability of AGG, there is also evidence for changes in AGG with age. Parents' ratings of AGG showed a decline in mean scores of AGG from age 4 to 18 years (Stanger *et al.,* 1997; Tremblay, 2002). This is especially true in the ages from preschool to elementary school years, when the prevalence of AGG declines (Kingston and Prior, 1995).

Using a developmental twin study approach, we can extend prior studies by determining how genetic and environmental processes are involved in the stability

[1] Department of Biological Psychology, Vrije Universiteit, Amsterdam, The Netherlands.

[2] Department of Psychiatry and Medicine (Division of Human Genetics), Center for Children, Youth and Families, and University of Vermont, College of Medicine, Burlington, Vermont.

[3] To whom correspondence should be addressed at Vrije Universiteit, Department: Biological Psychology, Van der Boechorststraat 1, 1081 BT Amsterdam, The Netherlands. Tel: +31 (0)20-4448949; Fax: +31 (0)20-4448832. e-mail: toos@psy.vu.nl

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and change of AGG over the course of development. Cross-sectional twin designs (e.g., studies of children at successive ages) can give absolute estimates of the genetic influences on AGG at different ages, yet provide no information about the change and interaction of genetic and environmental influences across development. By employing longitudinal designs, it becomes possible to investigate the ways in which both genes and environment contribute to the processes of stability and change across development.

Twin Studies of Aggressive Behavior

Using the twin method, clear genetic influences on AGG are established (Bergeman and Seroczynski, 1998). Hudziak *et al.* (2003, this issue) report that genetic contributions to AGG account for roughly 65% of the variance regardless of the age or sex of the subject or the type of informant. In a meta-analysis of AGG, Miles and Carey (1997) reported an overall genetic effect that accounts for 50% of the variance in aggression. In addition they reported that heritability estimates varied by age. In youth (18 years or younger), both genes and shared environment were important, but for adults only genes played a role. A recent study that conducted a meta-analysis to estimate the heritability on antisocial behavior also reported age differences (Rhee and Waldman, 2002). They compared heritabilities for children (below age 13), adolescents (age 13–18), and adults (above age 18). For all three age-groups, both genetic and shared environment factors were important, but the magnitude of the familial influences decreased with increasing age. These research findings suggest that the heritability of AGG depends on the age of the subjects studied, but they provide no information about how genes and environment may affect the processes of stability and change of AGG. Drawing conclusions from the existing longitudinal studies about the genetic stability of AGG is limited by the complexity of the results because they differ by assessment instruments, the definition of aggression, the age of the subjects, and the length of the intervals. McGue *et al.* (1993) assessed the stability of AGG, using the Multidimensional Personality Questionnaire (MPQ), in a young adult sample. Over a 10-year period AGG showed a stability of 0.54 and was accounted for largely by genetic factors. The genetic correlation between the two time points was 0.75 and pointed to large continuity of the same genetic influences for AGG. Using the Child Behavior Checklist (CBCL) Schmitz *et al.* (1995) examined the genetic influences on AGG when twins were 2 and 7 years old. For AGG they found 100% of overlap in genetic influences. However, these results should be interpreted with care because the study was conducted in a very small sample. van der Valk *et al.* (in press) collected maternal ratings of the CBCL for 3873 twin pairs at age 3 and four years later for 1924 twin pairs at age 7. In this study they reported on the Externalizing Behavior Scale, a scale that included both Aggressive Behavior and Rule-Breaking Behavior. Between age 3 and 7 the phenotypic stability of 0.54 could be for 55% explained by the genetic factors. The overlap in genetic influences was 50%. Recently, the study was extended with assessments of problem behavior at ages 10 and 12 (Bartels *et al.,* submitted [b]). The number of twin pairs at ages 3, 7, 10, and 12 were, respectively, 5602, 5115, 2956, and 1481. From age 3 the 4-, 7-, and 9-year phenotypic stability for externalizing behavior was respectively 0.55, 0.49, and 0.48. From age 7, they reported a 3-year phenotypic stability of 0.73 and 5-year stability of 0.68. The influence of genetic factors on the stability differed between girls and boys. On average, genetic factors explained 67% of the stability over the years for boys and 53% for girls. New genetic influences emerged at all four ages, but were most evident between age 3 and 7. Research findings on antisocial behavior also showed large genetic stability. For a group of 405 adolescent twins and their siblings, a stability of 0.63 was observed for antisocial behavior over a 3-year period (O'Connor *et al.,* 1998). More than 50% of this stability could be accounted for by the same genetic factors. van den Oord and Rowe (1997) analyzed antisocial behavior on three occasions in middle childhood (ages 4–6, 6–8, and 8–10) and obtained the same kind of results. The stability of antisocial behavior was entirely explained by genetic factors and shared environmental factors. Unique environmental factors contributed only to change of problem behavior. Recently, Jacobson *et al.* (2002) examined the sex differences in the underlying genetic and environmental architecture of the development of antisocial behavior from childhood to adulthood. They reported that the overlap in genetic influences between childhood and later ages was small. In contrast, they report similar environmental factors affecting antisocial behavior from childhood to adulthood. In summary, the general genetic factors contribute to a large extent to the stability of AGG and related problem behavior despite large differences in methods, measures, and ages.

The main goal of this paper is to examine how genetic and environmental processes are involved in the stability and change in AGG from early childhood to adolescence, as measured by parental reports on the

Child Behavior Checklist (CBCL). As in the case of genetic and environmental factors, developmental processes themselves cannot be measured directly, but can be inferred by applying statistical models. The common factor model and the simplex model are two frequently used statistical models to represent two kinds of developmental mechanisms (Lemery *et al.,* 1999). A common factor model represents a developmental mechanism that suggests one stable underlying factor that influences behavior at each age. The stability of AGG across ages is completely explained by the same factor. In this model, developmental changes could be reflected in changing factor loadings across time. It is also possible to account for change by including age-specific effects. In a simplex model the successive levels of behavior are causally linked so that each new event or change builds upon earlier experiences. Here, stability is explained as the part of earlier influences and experiences that are transmitted to subsequent ages. In addition to the effects of past behavior, new influences may enter at each age to account for change. After entering, the effect of these new influences may be transmitted to later time points.

In a longitudinal study the two developmental models can be distinguished because their underlying development mechanisms will result in a different pattern of correlations. A simplex model implies that measures taken closer in time are more highly correlated than measures further apart in time. Thus the correlation will decrease with increasing length of time between time points and the correlation matrix will take the form of a simplex (Guttman, 1954). In contrast, the common factor model assumes no specific correlation pattern as a function of time.

In genetic research the two longitudinal models may be used to make inferences about the underlying genetic and environmental effects of stability and change (Boomsma and Molenaar, 1987; Pedersen, 1991). Both models allow us to assess the relative importance of genetic and environmental factors at each time point. In addition, the models can test specific age-related changes in genetic and environmental influences. Important questions are: Do the same genetic and environmental influences operate throughout the development? Or: Are there "new" genetic and environmental influences that operate at specific points in time? Both models can be used to answer these kinds of questions, but the common factor model implies that the same genetic and shared environmental factors, for example, adverse home upbringing, underlay the stability of AGG on a constant manner through the development. The simplex model implies a more dynamic process with an age-to-age transmission of genetic and/or environmental effects and with age-specific effects. New influences may be due to "new" genes that may turn on at a specific age to regulate specific biological processes or to new environmental experiences, such as new peer groups or new school experiences.

In the present study, longitudinal twin data of the CBCL as reported by the mother were used to study the underlying developmental processes of AGG in childhood. Maternal reports on AGG were obtained for 6488 three-year-old twin pairs, 5475 seven-year-old twin pairs, 2983 ten-year-old twin pairs, and 1509 twelve-year-old twin pairs. By using structural equation modeling techniques, we examined the genetic and environmental contribution to stability and change of AGG. Because the data were collected for an equal number boys and girls, we could investigate whether the observed stability could be explained by the same pattern of genetic and environmental influences in boys and girls.

METHOD

Sample

The data of the present study are derived from a large ongoing longitudinal study, which examines the genetic and environmental influences on the development of problem behavior in families with 3- to 12-year-old twins. The families are volunteer members of the Netherlands Twin Register (NTR), kept by the Department of Biological Psychology at the Free University in Amsterdam (Boomsma *et al.,* 2002). From 1986 the NTR has recruited families with twins a few weeks or months after birth. Currently 40–50% of all multiple births are registered by the NTR. For the present study, we included data of 3- and 7-year-old twin pairs from cohorts 1986–1994, of 10-year-old twin pairs from cohorts 1986–1991, and of 12-year-old twin pairs from birth cohort 1986–1989. Parents of twin pairs were asked to fill in questionnaires about problem behavior for the eldest and youngest twin at ages 3, 5, 7, 10, and 12 years. After 2 months a reminder was sent to nonresponders, and after 4 months those who still did not respond were telephoned. This procedure resulted in a response rate (at least one questionnaire was returned) of 77% for age 3. From age 3 to 7, age 7 to 10, and age 10 to 12, the participation was continued by 80%. Nonresponders also include twin families who changed addresses. There were also families who did not participate at one age but entered the study again at subsequent ages.

For 822 same-sex twin pairs, zygosity was based on blood group ($n = 424$) or DNA polymorphisms ($n = 398$). For the remaining twins, zygosity was determined by questionnaire items about physical similarity and frequency of confusion of the twins by family and strangers (Goldsmith, 1991; Rietveld *et al.,* 2000) obtained at age 3, 7, 10, and 12 years. The classification of zygosity was based on a discriminant analysis, relating the questionnaire items to zygosity based on blood/DNA typing in a group of same-sex twin pairs. According to this analysis, the zygosity was correctly classified by questionnaire in nearly 95% of the cases. If a discrepancy in zygosity status appeared across ages, then the most frequent zygosity status was used.

A family was excluded if one of the twins had a disease or handicap that interfered severely with normal daily functioning (about 2%). Table I gives an overview of the number of families participating in the present study. For 4973 twins pairs, CBCL data were available for two ages (3 and 7 years); for 2459 twin pairs, CBCL data were available for 3 ages (3, 7, 10 years); and for 1150 twin pairs, the data were available for all four assessments. As shown in Table I, the sample size decreases by age (3, 7, 10, and 12 years) because this is an ongoing longitudinal study in which we add new cohorts annually. Therefore, for a large part of the 3-year-old sample, we have no longitudinal data for the reason that they did not yet reach the age of 7, 10, or 12 years.

Socioeconomic status (SES) was obtained from a full description of the occupation of the parents when the children were 3 years of age. The level of occupation was coded according to the system used by Statistics Netherlands (CBS, 1993). The code was based on the mental complexity of the work and ranged from low skilled to scientific work. An earlier comparison of the parental SES distribution with those obtained for the general Dutch population showed a slightly lower frequency of the lower-SES groups (Rietveld *et al.,* submitted). The percentages were 24% and 32% (low SES), 47% and 40% (middle), and 29% and 28% (high) for the twin sample and the Dutch population, respectively. Thus there is only a small discrepancy.

Table I. Number of Participating Twin Pairs at Each Age

	Age 3	Age 7	Age 10	Age 12
MZM	1055	927	526	289
DZM	1066	898	471	237
MZF	1226	1069	621	317
DZF	997	858	458	233
DOS	2144	1723	907	433
Total	6488	5475	2983	1509

Note: MZM = Monozygotic males; DZM = dizygotic males; MZF = monozygotic females; DZF = dizygotic females; DOS = dizygotic opposite sex.

Rating Scale

Behavior Problem Rated by the Parents

At age 3, behavior was measured with the CBCL/2-3, a questionnaire that included 100 items that describe specific behavioral, emotional, and social problems. Parents were asked to rate the behavior that the child displayed currently or in the past 2 months on a 3-point scale: 0 if the problem item was not true, 1 if the items was somewhat or sometimes true, and 2 if it was very true or often true. The AGG syndrome is constructed for the Dutch population (see Koot *et al.,* 1997) and is compatible with the syndrome scale as developed by Achenbach (1992). The AGG syndrome derived form the CBCL/2-3 contains nine items.

At ages 7, 10, and 12 years, problem behavior was measured with the CBC/4-18 (Achenbach, 1991), a questionnaire of 113 items developed to measure problem behavior in 4–18 year-old children. Again parents were asked to rate the behavior of the child in the preceding 6 months on a 3-point scale. The AGG syndrome derived from the CBCL/4-18 contains 20 items and partially overlaps with the CBCL/2-3. The items of the AGG syndrome of both questionnaires are given in the paper of Hudziak *et al.* (this issue).

Statistical Analyses

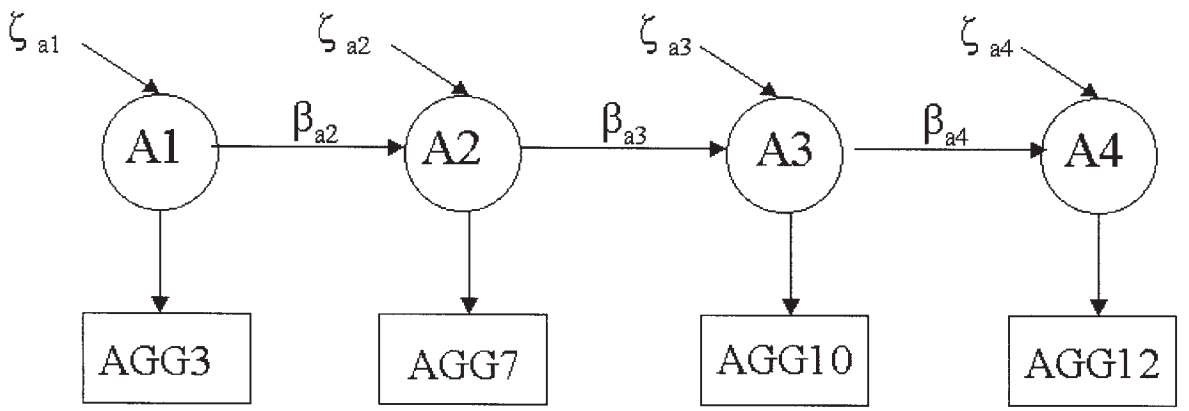
Phenotypic Stability

The phenotypic stability coefficients, the twin and the twin-cross correlations were calculated by using the statistical software program Mx (Neale *et al.,* 1999). A fully saturated model was fitted to the data, in which all variances, covariances, and means were estimated in each zygosity group, without any constraints. In the next step, the stability coefficients were constrained to be equal for MZ and DZ twins, and for oldest and youngest twins. This resulted in six stability coefficients for each sex. To test sex differences, the coefficients were constrained to be equal across boys and girls, and the fit of the model was compared with the fit of the previous model. Accordingly, we tested whether the pattern of phenotypic correlations was best described by a simplex model or by a common factor model.

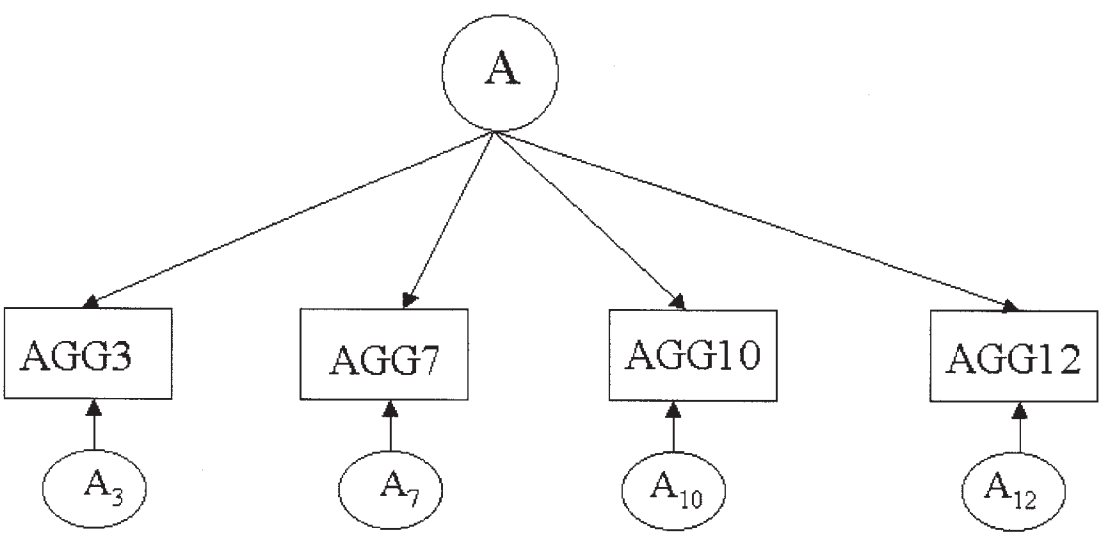
Genetic Analyses

First, the twin cross-correlations were calculated (e.g., AGG at age 3 in the first-born twin with AGG at age 7 in the co-twin). The cross-correlations provide a

first indication of the importance of genetic and environmental influences on the stability of a trait (Neale and Cardon, 1992). If a cross-correlation is higher for MZ than for DZ twins, genetic factors influence the stability of the trait. Next, we employed longitudinal genetic developmental models to investigate the contribution of genetic and environmental factors on the stability of AGG across the ages 3, 7, 10, and 12 years. In univariate genetic analyses the variance of a trait is decomposed into genetic and environmental components; in longitudinal analyses the association between assessments over time is decomposed into a genetic and a nongenetic part. By comparing the cross-twin, cross-variable covariance in MZ and DZ twin pairs, the covariance of two or more time points can be decomposed into its genetic and environmental components. We employed two developmental models that may explain the pattern of correlations among time points. The common factor model (also called an independent pathway model) contains one latent genetic (environmental) factor that accounts for the covariance among the four assessments. In addition to this common factor, the model includes unique genetic (environmental) factors that are specific for each assessment. An example of this model is given in Figure 1. The second model is a simplex model and includes causal pathways between



A) Simplex model



b) Common factor model

Fig. 1. Path diagrams of the models used to investigate the underlying developmental mechanism of aggressive behavior across four time points (AGG3, AGG7, AGG10, AGG12). (a) A simplex model, and (b) a common factor model with age-specific effects. In the simplex model, A1–A4 refers to additive genetic factors, parameter β*i* is the transmission parameter, and ζ*i* denotes the effect of novel influences specific for each age. In the common factor model, A represents the common factor and A_3, A_7, A_{10}, A_{12} refer to the effects specific for age 3, 7, 10, and 12 years, respectively. Instead of the A factor, the shared environmental factors (C) or nonshared environmental factors (E) could be substituted in the models.

genetic (environmental) factors at adjacent occasions (Figure 1). As a result, genetic (environmental) factors at a particular age are influenced by factors proceeding that age. Such relationships amongst latent factors are termed autoregressive. Furthermore, the model included innovations (Neale and Cardon, 1992). The innovation is part of the latent factor at an assessment that is not caused by a latent factor at a preceding assessment. At the first assessment the first latent factor cannot be explained by factors associated with an earlier point in time, and therefore this factor itself is regarded as an innovation. The simplex model is most suitable to longitudinal series in which there is occasion-to-occasion transmission and correlations decrease with increasing distances between time points (Boomsma and Molenaar, 1987; Pedersen, 1991). To test the fit of the two developmental models, the Cholesky model was used as reference model. In the Cholesky model the number of factors equals the number of observed variables. The first factor contributes to all four assessments; the second factor influences the subsequent three assessments and so on. The model is a fully saturated unconstrained model and provides a general description of the contribution of genetic and environmental factors to the covariances. Because results from previous genetic analyses of the same twin sample (Hudziak *et al.,* this issue) suggested the influence of additive genetic factors, shared and nonshared environmental factors, the longitudinal models included these three sources of variation. Prior to the genetic analyses, the developmental models were evaluated at the phenotypic level.

The different models were fitted to the data with MX (Neale *et al.,* 1999), by the method of raw maximum likelihood estimation. Using raw data, Mx provides the possibility to handle missing data and allowed us to retain twin pairs who have missing data at one or more assessments. In this procedure the likelihood is calculated separately for each pedigree and the product of these likelihoods (i.e., the sum of the log-likelihood) is maximized. The use of maximum likelihood estimation required that the data were approximately normally distributed. Therefore the data were square-root transformed.

Goodness of fit of the longitudinal models was assessed by likelihood-ratio χ^2-tests. Nested models were compared by using the likelihood ratio chi-square tests, which used the differences between −2-log likelihood of the full model from that of a restricted model. This difference is distributed as a χ^2. The degrees of freedom (df) for this test are equal to the difference between the number of estimated parameters in the full model and that in a restricted model. Because the simplex and common factor models are not nested, it is not possible to use the likelihood-ratio χ^2-test to compare whether one model fits better. To select the best model the Akaike's Information Criterion (AIC) was used. The AIC is functionally related to the chi-square minus twice the degrees of freedom (Akaike, 1987). The model with the lowest value is chosen as the best model.

RESULTS

Sample

Because selective attrition may bias estimates of genetic and environmental influences (Heath *et al.,* 1998), we tested whether the baseline characteristics differed between continuing and noncontinuing participants. A prior study that used almost the same sample showed that nonparticipants reported slightly more attention problems at the baseline assessment (age 3 years) (Rietveld *et al.,* in press). We examined whether AGG at age 3 (the baseline) differed between continuing and noncontinuing participants. The continuing participants were divided into three groups, those who participated at ages 3 and age 7; those who participated at ages 3, 7, and 10; and those who participated all four times. The noncontinuing participants consisted of persons that participated only at age 3. The aggression score was randomly selected from the oldest or the youngest of a twin pair. At age 3, the mean AGG score was 3.38 for the noncontinuing group. For the other groups, participating two, three, and four times, the mean scores were 3.35, 3.22, and 3.13, respectively. However, these scores did not differ significantly among the four groups [$F(3,6657) = 2.38$, $p = 0.68$]. In addition, using multivariate logistic regression, we tested whether educational level of the mother, aggressive behavior at age 3, or socioeconomic level influenced the participation rate at age 7. The results showed that the willingness to participate at age 7 depended slightly on SES. Families with a lower socioeconomic status were less likely to continue to participate at the age 7 (odds ratio = 1.1, 95% confidence interval 1.00–1.22).

These results are important in the light of the validity of the raw maximum likelihood procedure. This procedure depends upon model assumptions about the pattern of missing data mechanism. In general, a distinction is made between three types of missing data mechanism (Little and Rubin, 1987). First, the missing data can be classified as missing completely at random (MCAR), when the probability of missing data does not depend on the observed and unobserved data. Second, missing data can be missing at random (MAR), when

the probability of the missing data depends on the observed data but not on the unobserved data, given the observed data. Third, the pattern is missing ignorably (MI) when the probability of nonresponse depends on unobserved data. Raw ML procedures may yield consistent estimates if the data is MCAR or MAR (Wothke, 2000). It is tenable that the pattern of missing data in our study is at least MAR and that for some variables the pattern is even MCAR.

Phenotypic Stability

In Table II the phenotypic stability coefficients of AGG for each follow-up interval are given separately for boys and girls. For both boys and girls, the stability coefficient was moderate between ages 3 and 7, but the coefficients were higher for the shorter time intervals. If the stability coefficients were constrained to be equal across sex, the fit of the model deteriorated significantly ($\Delta\chi^2 = 15.437$, $\Delta df = 6$). This means that stability coefficients are different for boys and girls. As shown in Table II, the sex differences were very small and were most prominent in the older age-groups.

The pattern of coefficients gives us a first indication of the underlying developmental pattern of AGG. The stability coefficients were largest for the smaller time intervals and decreased as the time between assessments increased. This pattern suggests a simplex model. Prior to the genetic analysis, we tested which developmental mechanism gives the best fit to the data at the phenotypic level. As shown in Table III, for both boys and girls the simplex model provided the best fit.

Table II. Phenotypic Correlations, Twin Correlations (MZ, DZ, and DOS) and Twin Cross Correlations for CBCL AGG Syndrome of Boys (below diagonal) and Girls (above diagonal)

Boys \ Girls	Age 3	Age 7	Age 10	Age 12
Phenotypic				
Age 3	1	.48	.43	.41
Age 7	.48	1	.69	.66
Age 10	.43	.73	1	.71
Age 12	.42	.67	.77	1
MZ				
Age 3	.81/.82	.45	.41	.40
Age 7	.47	.83/.84	.62	.60
Age 10	.43	.66	.84/.79	.64
Age 12	.42	.62	.69	.86/.83
DZ				
Age 3	.55/.53	.34	.31	.30
Age 7	.33	.48/.53	.42	.39
Age 10	.31	.37	.50/.55	.41
Age 12	.27	.31	.37	.45/.55
DOS				
Age 3	.48			
Age 7	.29	.51		
Age 10	.28	.38	.47	
Age 12	.29	.38	.40	.57

Note: MZ = Monozygotic; DZ = dizygotic; DOS = dizygotic opposite sex.

Genetic Analyses

Table II presents the twin correlations (on the diagonal) and twin cross-correlations for AGG for same-sex MZ and DZ twin pairs and for opposite-sex twin pairs. The cross-correlations are used as a first indication of the involvement of genes and environmental factors on stability across ages. At all ages, the MZ cross-correlations were larger than the DZ cross-correlations and suggested genetic influences on the stability across ages. In addition, the DZ cross-correlations were larger than expected on the basis of genetic influences alone, and therefore shared environmental influences seemed also to contribute to the stability. In both the MZ and DZ twin groups, the cross-correlations decreased as a function of time, but the decrease was less than expected based on the simplex model alone. The higher twin cross-correlations could also suggest that both a simplex and a common factor mechanism operate, but that one mechanism operates at the genetic level and the other operates at the environmental level.

Table III. Model Fitting Results of Phenotypic Developmental Models for CBCL Aggression

Model	−2ll	df	Compared to model	$\Delta\chi^2$	Δdf	p	AIC
Boys							
1. Fully saturated	29645.681	11021	–	–	–	–	–
2. Common factor	29691.682	11023	1	46.001	2	0	42.001
3. Simplex	**29647.028**	**11023**	**1**	**1.347**	**2**	**0.51**	**−2.653**
Girls							
1. Fully saturated	29971.992	11262	–	–	–	–	–
2. Common factor	29993.978	11264	1	22.058	2	0	18.058
3. Simplex	**29972.307**	**11264**	**1**	**0.315**	**2**	**0.85**	**−3.685**

A series of model fitting analyses was conducted to test which developmental model best described the data. For each variance component (A, C, and E) tests were performed to determine whether a common factor model or a simplex model provided the best model in explaining the data. We examined the goodness-of-fit of the two developmental models for each variance component (A, C, and E) separately. This was done by testing the common factor or the simplex model for one variance component while the other variance components were expressed as Cholesky decompositions. A model, in which all variance components were expressed as a Cholesky, was taken as reference for evaluating changes in χ^2 and associated degrees of freedom. In all models the estimates of the parameters were allowed to differ across sex. Table IV presents the fitting results of the various models. For the genetic influences, both the common factor model and the simplex model provided a better fit than the Cholesky model. On basis of the lower AIC value, the simplex model was the preferred developmental model. For the shared environmental influences, a common factor model provided a better fit. For the nonshared environmental influences, neither the simplex nor the common factor model provided an adequate fit. The model with a simplex structure for the additive genetic influences, a common factor for shared environmental influences and a Cholesky for nonshared environmental influences was chosen as final model. The fit of this model provided a better fit than the fully saturated model.

To test whether the parameter estimates differed between boys and girls, the parameters of the final model were constrained to be equal across sex, and the fit of the model was compared with the model without sex differences. The fit of a model without sex differences deteriorated significantly, which means that the magnitude of the genetic and environmental influences differed between boys and girls. However, inspection of the parameter estimates of boys and girls in Figure 2 reveals only small differences. The significant sex differences were most likely due to variance differences between boys and girls (especially after age 7). The variances of the boys' transformed AGG score are 0.73, 1.43, 1.58, and 1.54 for, respectively, age 3, 7, 10, and 12, and for girls the variances were 0.71, 1.45, 1.31, and 1.33 for, respectively, age 3, 7, 10, and 12. For an overview of the variances for each zygosity see Hudziak *et al.* (this issue).

The unstandardized estimates, as shown in Figure 2, can be used to compute the relative contribution of G and E to the age-specific total variances and stability coefficients. The genetic contribution to AGG at each age consisted of genetic influences novel to that age (=innovation effect), plus the genetic effects that were already operating at a previous age (transmission). The standardized innovation effect is achieved by dividing the squared innovation coefficient by the total variance (the total variance of each age is calculated as the sum of all genetic and environmental variance components). For example, the innovation effect at age 7 for boys is $(0.832)^2/1.43 = 48\%$. The percentage of the genetic variance at age 7 that is transmitted from age 3, can be obtained by dividing the product of the squared transmission coefficient and the genetic variance of

Table IV. Model Fitting Results of Longitudinal Developmental Models for CBCL Aggression

Model	−2ll	df	Compared to model	$\Delta\chi^2$	Δdf	p	AIC
1. Saturated model A: Cholesky C: Cholesky E: Cholesky	78977.640	32791	–	–	–	–	–
2. A: Simplex	78986.673	32797	1	9.03	6	0.17	−2.967
3. A: Factor structure	78986.346	32795	1	8.71	4	0.07	0.706
4. C: Simplex	78989.428	32797	1	11.79	6	0.07	−0.212
5. C: Factor structure	78981.358	32795	1	3.72	4	0.45	−4.282
6. E: Simplex	79004.664	32797	1	27.02	6	0.0	15.024
7. E: Factor structure	78987.158	32795	1	9.52	4	0.05	1.518
8. Final model A: Simplex C: Factor E: Cholesky	78991.93	32801	1	14.29	10	0.16	−5.71
9. Final model, no sex differences	79051.92	32826	8	59.97	25	0.0	9.972

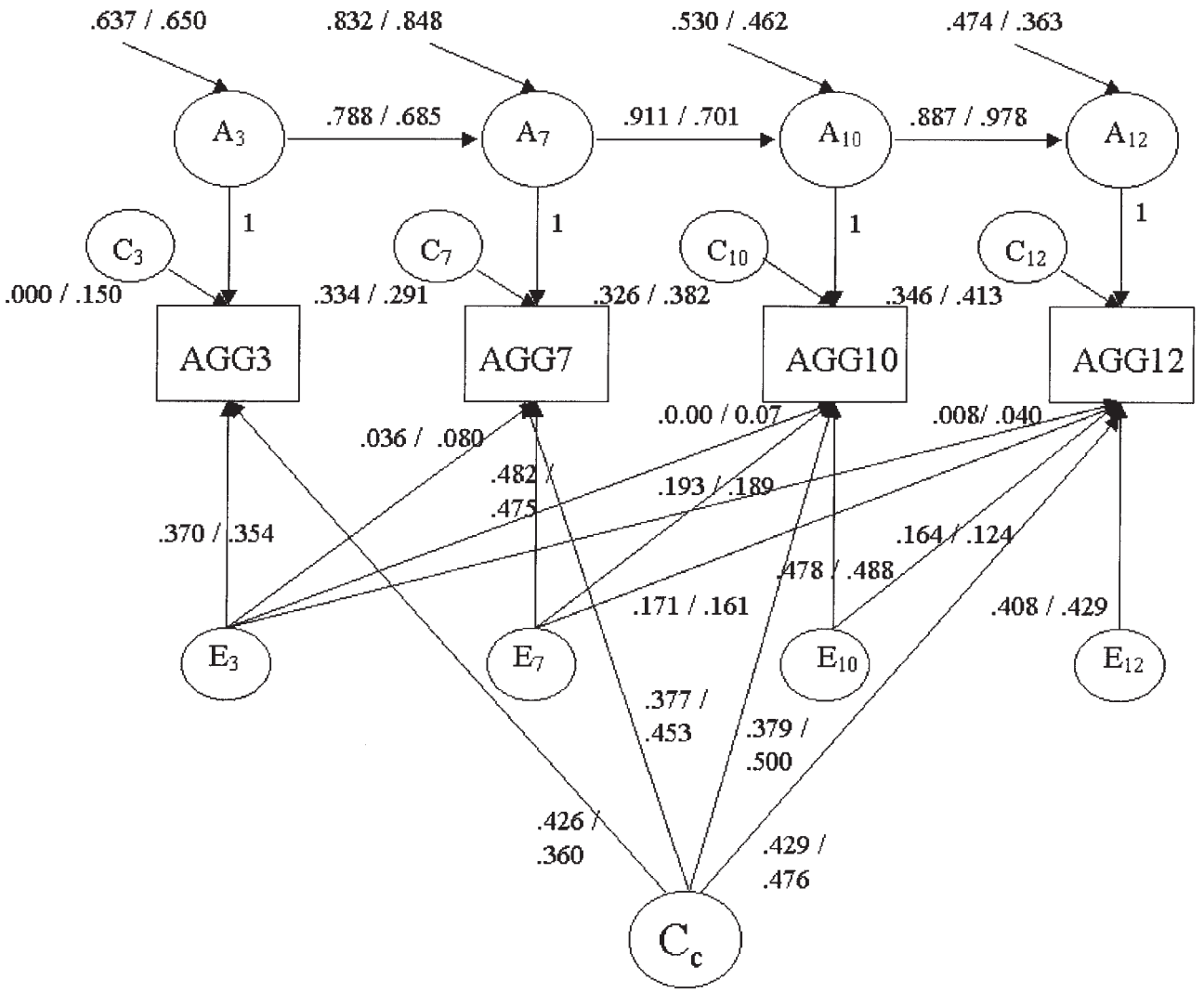


Fig. 2. Unstandardized estimates of the final model for aggressive behavior (AGG) from age 3 to age 12 separately for boys and girls (boys/girls). The squares represent the observed variance for each age (AGG3, AGG7, AGG10, AGG12). The circles represent the latent factors. For additive genetic factors (A_3, A_7, A_{10}, and A_{12}) a simplex model is included. For the shared environmental effects a common factor (Cc) and 4 age-specific factors (C_3, C_7, C_{10}, C_{12}) are included. The model includes a Cholesky decomposition for nonshared environmental factors (E_3, E_7, E_{10}, E_{12}).

the previous time point by the total variance: $(0.788)^2 * 0.41/1.43 = 18\%$. The contribution of shared environmental factors to the total variance of AGG at a specific age consisted of age-specific shared environmental influences and of environmental influences shared with all ages. For boys at age 7, the contribution of the specific shared environmental influence is calculated as $(0.334)^2/1.43$ (= total variance) = 8%, and the contribution of the common factor is $(0.377)^2/1.43 = 10\%$. An overview of the relative contribution of G and E for each age is given on the diagonal of Table V.

At each age, the contribution of genetic factors was large and remained quite stable across ages. Averaged over ages, 64% (for boys) and 57% (for girls) of the variance was explained by genetic factors. The contribution of age-specific genetic factors differed across ages. At age 7 years, about 50% of the total variance consisted of new genetic variance. After age 7, only a small part of the variance consisted of new genetic variance. The contribution of shared environmental influences to the total variance was around 20% for boys and around 25% for girls. Shared environmental variance at each age is the sum of variance due

Table V. Relative Contributions of Genetic and Environmental Factors to the Total Variance and Covariances of AGG in Boys and Girls

	Additive genetic				Shared environment				Nonshared environment			
	Age 3	Age 7	Age 10	Age 12	Age 3	Age 7	Age 10	Age 12	Age 3	Age 7	Age 10	Age12
Boys												
Age 3	.56 (.47–.64)				.25 **[.25 + *0*]** (.20–.33)				.19 (.17–.21)			
Age 7	.65 (.55–.75)	.66 **[.18 + *.48*]** (.59–.72)			.33 (.22–.44)	.18 **[.10 + *.08*]** (.12–.24)			.02 (0.–.05)	.16 **[0 + *.16*]** (.15–.18)		
Age 10	.64 (.50–.77)	.78 (.71–.85)	.67 **[.49 + *.18*]** (.60–.74)		.36 (.23–50.)	.13 (.07–.21)	.16 **[.09 + *.07*]** (.10–.23)		0 (0.–0.2)	.08 (.06–.10)	.17 **[.03 + *.14*]** (.15–.19)	
Age 12	.58 (.45–.71)	.76 (.67–.83)	.78 (.69–.84)	.67 **[.53 + *.14*]** (.58–.75)	.41 (.28–.54)	.16 (.09–.24)	.13 (.07–.21)	.19 **[.12 + *.08*]** (.12–.28)	0.0 (0–.04)	.08 (.06–.11)	.09 (.06–.12)	.14 **[.04 + *.10*]** (.12–.17)
Girls												
Age 3	.60 (.55–.69)				.22 **[.19 + *.03*]** (.13–.30)				.18 (.16–.19)			
Age 7	.60 (.48–.71)	.64 **[.14 + *.50*]** (.56 –.71)			.34 (.23–.40)	.20 **[.14 + *.06*]** (.13–.27)			.06 (.04–.08)	.16 **[0 + *.16*]** (.15–.18)		
Age 10	.50 (.44–.63)	.67 (.56–.76)	.50 **[.34 + *.16*]** (.40–.59)		.44 (.40–.57)	.23 (.14–.33)	.29 **[.19 + *.10*]** (.26–.36)		.06 (.03–.09)	.10 (.08–.12)	.21 **[.03 + *.18*]** (.19–.24)	
Age 12	.52 (.38–.66)	.68 (.57–.78)	.66 (.55–.77)	.55 **[.46 + *.09*]** (.44–.66)	.44 (.30–.47)	.23 (.14–.24)	.24 (.19–.35)	.29 **[.17 + *.12*]** (.18–.39)	.04 (0–0.08)	.09 (.06–.12)	.10 (.07–.13)	.16 **[.03 + *.14*]** (.14–.19)

Note: On the diagonals the effects of A, C, and E are partitioned into stability and time specific effects (between squared brackets and in bold type). Genetic influences are partitioned into transmitted (first number) and innovation effects (second number and in italics). Shared and nonshared environmental influences are partitioned into common (first number) and age-specific effects (second number and in italics). The 95% confidence intervals of the estimates are given in round brackets.

to one shared factor plus the variance specific for each age. For boys, the contribution of age-specific shared environmental factors to the total variance was around 8% for each age. For girls, the contribution of age-specific shared environmental factors tended to increase with age. The nonshared environmental variance consisted mainly of unique, age-specific variance.

Also illustrated in Table V is the contribution of genes and environment to the stability of AGG. Here, the emphasis is on the extent to which the phenotypic correlation between two time points can be explained by genetic or environmental influences. In the off-diagonals of Table V, the proportion of the phenotypic correlation that is due to the genetic and environmental influences is reported. The stability was mainly determined by genetic and shared environmental factors. For boys, genetic factors accounted on average for 69%, and shared environmental factors accounted for 25% of the phenotypic stability across ages. In girls, the contribution of genetic influences to the stability was somewhat smaller (60%), and the contribution of shared environmental influences was somewhat larger (32%). For both boys and girls, the contribution of nonshared environmental factors to the stability was small and had mainly age-specific effects.

Table VI presents the correlations among the genetic and environmental factors. The genetic and environmental correlations describe the extent to which the same genes or environmental factors contribute to the phenotypic correlation of AGG at two time points. A high correlation indicates that the same genetic influences operate at each of the two time points. The pattern of genetic correlations reflected a simplex pattern, in which the overlap between genetic influences is larger for assessments closer in time (especially for ages 7, 10, and 12) than between assessments with longer time intervals (that is, the correlation among age 3 and age 7, 10, and 12). At ages 7, 10, and 12 the overlap in genetic factors is around 80%. Only 50% of the genetic factors that impact AGG at age 3 have an impact at age 7. The pattern of shared environmental correlations indicated that the same shared environmental influences were important from early childhood to adolescence. The somewhat lower correlations among the shared environmental factors after age 7 suggested more changes in this period. The unique nonshared environmental correlations were small and indicated that their influence was mainly age-specific. After age 7, the factor correlations were somewhat larger, indicating that some nonshared environmental influences contributed to stability.

DISCUSSION

In the present study two developmental models were tested to examine the genetic and environmental influences on the processes of stability and change of AGG from age 7 to 12 years. Both developmental models, the simplex and the common factor model, suggest stability, but the mechanism that accounts for the stability may differ (Cardon *et al.,* 1992). Our data indicate that during childhood AGG is a highly stable behavior, which is largely accounted for by genetic influences. The underlying developmental mechanism of genetic effects can be best described by a simplex model, which suggests a dynamic developmental process consisting of transmission of existing genetic effects interacting with new genetic influences. The contribution of new genetic influences was most prominent at age 7. The influence of shared environmental factors on the stability of AGG was modest and could be best described by a common factor model, implying the same shared environmental influences underlying the development of AGG from early childhood to adolescence. In addition to these relatively stable shared environmental influences, age-specific shared environmental influences appeared. The effect of nonshared environmental factors seemed mainly age-specific and had only a minimal contribution to the stability of AGG.

Table VI. Genetic and Environmental Factor Correlations Separately for Boys (above the diagonal) and Girls (below the diagonal) Based on the Final Model

	Additive genetic				Shared environment				Nonshared environment			
	Age 3	Age 7	Age 10	Age 12	Age 3	Age 7	Age 10	Age 12	Age 3	Age 7	Age 10	Age 12
Age 3	1.00	.52	.44	.39	1.00	.75	.76	.78	1.00	.07	0	.02
Age 7	.47	1.00	.86	.76	.78	1.00	.57	.58	.17	1.00	.37	.36
Age 10	.38	.82	1.00	.89	.73	.67	1.00	.59	.14	.38	1.00	.46
Age 12	.35	.75	.91	1.00	.70	.63	.60	1.00	.09	.35	.37	1.00

Stability and Change: Phenotypic Results

In agreement with several other studies (Koot, 1995; Loeber and Hay, 1997), AGG showed moderate to high stability from age 7 to 12 years. The stability coefficients ranged from 0.41 to 0.77 across varying intervals. As expected the stability coefficients decreased steadily with increasing time intervals. However, even after 9 years the stability coefficient was still 0.44 for boys and 0.36 for girls. The magnitude of the stability coefficients is consistent with the findings from a Dutch population study that assessed problem behavior of children in the same age range with the same assessment instrument (Verhulst and van der Ende, 1995). They reported for boys, ages 7–10 years, stability coefficients of 0.72, 0.66, and 0.48, respectively, for 4-, 6-, and 8-year intervals. In the present study, the stability coefficients for 7-year-old boys were 0.72 and 0.66, respectively, for 3- and 6-year intervals. Another finding in the study of Verhulst and van der Ende (1995) was that the stability coefficients were somewhat larger for the older (7–10 years) than for younger (4–10) children. Also, longitudinal results from the Pittsburgh Youth Study (Loeber and Hay, 1997) suggest an increase of the stability of aggression from age 6 forward. Indeed, we report greater stability coefficients in the older than in the youngest age-groups. The stability coefficient between age 7 and 12 was 0.66, and the stability coefficient between age 3 and 7 was 0.40 (for boys). These findings suggest that AGG becomes a more stable characteristic as children age. However, we cannot exclude that part of the explanation for lower stability measures between age 3 and 7 is due to differences in assessment instruments. At age 3, the scale AGG is derived from the CBCL/2-3, and is composed of 9 items, while from age 7 AGG is assessed with the CBCL/4-18, and this scale is composed of 20 items. Although the syndromes of both assessment instruments overlap with items, it can be questioned whether the syndromes measure exactly the same underlying construct at the different ages.

The Influence of Genetic Factors on Stability and Change

As suggested by the results in the cross-sectional study of AGG in the same sample of twin pairs, the magnitude of genetic and environmental influences on AGG was substantial and stable across age 3 to age 12 (Hudziak *et al.,* this issue). The present study extends the data of cross-sectional sample by applying genetic developmental models that could account for the stability during childhood and by testing whether the sources of genetic variation were the same at each age. The results showed that the stability of AGG was accounted for largely by genetic factors. Averaged over boys and girls, about 65% of the covariance could be explained by genetic factors. These findings are consistent with recent studies that found that stability of externalizing problems was explained largely by genetic factors (Bartels *et al.,* submitted [b]; van den Oord and Rowe, 1997; van der Valk *et al.,* in press).

The developmental mechanism of genetic factors was best described by a simplex model. This implies that the genetic influences operating at age 3 can exert their influence at later ages, but the influence diminished as the time between assessments increased. Meanwhile, there is room for new genetic influences. Thus the extent to which the same genetic and environmental factors operate during a developmental period may be different. In our data this is best illustrated by the genetic developmental change between age 3 and 7. At age 7 a large proportion of the genetic variance consisted of new genetic variance and only a small part of the genetic variance was transmitted from the previous age. The finding of genetic change in this period replicates the findings of recent genetic developmental studies that examined cognitive abilities and externalizing behavior in this life period (Bartels *et al.,* submitted [b]; Cardon *et al.,* 1992; van der Valk *et al.,* in press). Over this developmental period, children may experience many developmental transitions (i.e., on physical, cognitive, and social levels). It is the period in which children start with formal schooling and school-aged children may experience new environmental stressors. For example, for most of the children it is the first time that they participate in an organized peer group and have to form social relationships. These changing environmental influences, together with the accompanying changes in interactions between these new environmental influences and the biological make-up of the child, may influence the etiology of children's problem behavior (e.g., environmental influences on genetic risks).

In the period from age 3 to 7 years of age, important developmental changes take place; however, the period from age 7 to 12 years seems a reasonably stable period. Our results suggest no substantial genetic changes and support the idea that similar genetic influences affect AGG at age 7, 10, and 12. This pattern of stability fits well with the continuous type of antisocial behavior in Moffitt's theory (1993). According to this theory, two subtypes of antisocial behavior could be distinguished. One is called the continuous, "life-course-persistent" type, and the other type is called the

"adolescent-limited" type. A general finding is that the genetic influences are larger for the life-course-persistent type than for the adolescent-limited type (Eley *et al.,* 1999; Taylor *et al.,* 2000). The finding that AGG is a stable characteristic during childhood (at least from age 7) and the finding that it is a very heritable trait are consistent with the continuous type of Moffit's theory. We will extend these analyses as the twins age in order to investigate if new genetic or environmental variance emerges when the children become older. We will be especially interested to determine the transitional effects from 10 to 14 years of age, in the context of Moffitt's theory that predicts the rise of new environmental variance during puberty. Similarly, new genetic influences may arise in concert with the hormonal changes in puberty. As we begin our data collection on our twins at age 14, we will add a new informant, the twin himself/herself by collecting the Youth Self Report (YSR). The addition of the YSR will allow us to analyze data from multiple informants, across development, to determine if there are new genetic and environmental factors acting on AGG during the adolescent time period. We will combine the analyses of AGG with that of Rule Breaking Behavior (RB) (see Bartels *et al.,* this issue) to determine if the bivariate manifestation of AGG-RB is different in adolescents vs. younger-age children.

The Role of Environmental Factors in Stability and Change

About a quarter of the stability in aggressive behavior was accounted for by shared environmental influences, and the magnitude of this effect was quite stable across ages. Given that the common factor model was the best model for the shared environment, the results indicate that the same set of shared environmental influences exert their effect at each age. This is also supported by the pattern of the shared environmental factor correlations. Thus it seems that certain aspects of the family (SES, parental rearing practices, living in the same neighborhood, having overlapping social relationships) may be very important in the persistence of AGG in childhood. A large body of literature on familial risk factors for AGG exists. During preschool and childhood, familial factors such as lack of parental monitoring, parental aggression, permissiveness or inconsistency in discipline, and socioeconomic disadvantages promote aggressive behavior (Farrington, 1995). However, the effects of shared environment may have been inflated by parental bias. By using the same rater at two or more points, the prediction of AGG could to some extent reflect shared rater bias. The observed stability may reflect not only the stability of children's problem behavior but also the stability in the mother's perception of the child's development. Although this must kept in mind, previous studies on the CBCL have shown the effects of rater bias to be small, thus possible distortions are probably small (Bartels *et al.,* submitted [a]; Hudziak *et al.,* 2000; van der Valk *et al.,* 2001). Another factor that may explain a part of the shared environmental component is assortative mating. Assortative mating refers to nonrandom mating and may affect estimates of the heritability. Assortative mating raises the DZ correlation, while the MZ correlations remain unaffected because MZ twins are identical. In this way, assortative mating may bias the shared environmental component upward. Positive assortative mating of antisocial behavior was reported by Krueger *et al.* (1998).

In addition to the common shared environmental factor, age-specific shared environmental influences were found, which means that there is an environmental factor that makes twins more similar, but that its effect is age-specific. Because these age-specific environmental factors especially appeared at age 7, the period in which the children have already entered school, the most likely candidates for these influences are changing peer groups, changing teachers and accommodation of the school routine. The age-specific shared environmental factors could also reflect rater bias. It is conceivable that the mother modifies her view of certain problem behaviors when the children grow up.

Our findings showed that nonshared environmental factors operate mainly in a time-specific manner. Environmental factors that may promote aggression in one twin and not in the other can include illnesses, possible traumatic experiences, or specific school problems. After age 7, about 10% of the stability is accounted for by the nonshared environmental factors that are persistent over time. A good candidate for such effects may be the parental rearing style, which may be consistent across ages, but may be experienced differently by the twins (Feinberg and Hetherington, 2001). A number of studies have reported a relation between negative parental behavior and later AGG (Copeland *et al.,* submitted; Reiss *et al.,* 2000).

Sex Differences in Stability and Change

For this sample we reported sex differences reflecting higher scores for boys over girls and sex differences in levels of aggression that became more marked after the age of 7 (Hudziak *et al.,* 2003). Our

results indicated sex differences in the magnitude of genetic and environmental influences on AGG. At the same time sex differences in means became more pronounced, sex differences in genetic architecture appeared as well. After age 7, genetic factors have a larger effect for boys than for girls, whereas shared environmental influences were more important for girls than for boys. The same results were obtained for the stability of AGG. Apparently, disadvantaged familial factors are more important in the continuation of problem behavior in girls than in boys. These results agree with earlier reports of externalizing problem behavior in the same sample of twin pairs as used here (Bartels *et al.,* submitted [b]), and with the results of the meta-analysis of Miles and Carey (1997). The meta-analysis reported greater genetic influences on AGG in males than in females. However, results are inconsistent with some previous findings (Eley *et al.,* 1999; Jacobson *et al.,* 2000; Vierkko *et al.,* 2003). A direct comparison among studies is complicated by the use of different designs (e.g., cross-sectional vs. longitudinal), different measures, ages, or sample sizes. Possible explanations for the differences between our results and above mentioned studies are age differences and different definitions of AGG. As shown in previous research the etiology of individual differences of aggression and related behavior may be different and may vary across ages.

CONCLUSION

The findings of this study may be important to understand the causes of the developmental stability of AGG. It was shown that AGG was a highly stable characteristic and that most of the stability was explained by genetic factors. On average, genetic factors explained 65% of the stability. The extent to which the same genes contributed to the variance of AGG from age 3 to 12 years varied from 50% between age 3 and 7 to 91% between age 10 and 12. Shared environmental influences also influenced the stability, accounting for 27% of the covariance on average. The role of the shared environmental factors could be best described by a common factor model, implying a stable set of shared environmental factors underlying AGG at all ages. Parental bias and assortative mating may have inflated the estimates of shared environmental influences. Future research that includes data from multiple informants and parental data may be helpful to disentangle the effects of parental bias and assortative mating and real shared environmental influences.

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